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Associative Interference and Premorbid Adjustment in Schizophrenia

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ASSOCIATIVE INTERFERENCE AND PREMORBID ADJUSTMENT IN SCHIZOPHRENIA

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Life

Clifford Leo Hirsch was born in Milwaukee, Wisconsin, May 27, 1942. He was graduated from Washington High School in Milwaukee, January, 1961. He began his undergraduate studies at Marquette University, Milwaukee, and obtained a Bachelor of Science degree in June, 1965.

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Introduction

Associative Interference in Schizophrenia

Bleuler (1950) appears to have been one of the first to focus on associational disturbance as an explanation of the psychological deficit found in schizophrenia. Mednick (1958) elaborated on this concept and delineated the relationship between arousal level and associative disruption in schizophrenia. He anchored his thinking in the Hullian notion of generalized drive (D) which in Hull's system is the central motivational force (Hull, 1943). According to Hull a multiplicative relationship holds between response strength and drive. Increasing drive (D) raises the strength of all responses in the organism's response repertory because this type of general arousal is without direction. Mednick reasoned that in individuals with high drive levels learning of simple tasks (those with few competing responses) should proceed at a rapid rate. However, in complex tasks, learning should be impeded by response competition.

Mednick assumed that schizophrenics are individuals with very high drive levels, especially during the acute phase of their disorder. Although he did not elaborate on the etiology of heightened arousal, he felt that individuals in such a state effect an adjustment to the environment

which reduces stimulation (i.e., social and emotional withdrawal) and keeps drive within acceptable limits. However, some traumatic event interferes with this adjustment raising the drive level. With increased drive, stimulus and response generalization increase. The individual then becomes caught in what Mednick termed the "reciprocal augmentation of anxiety and stimulus generalization (p. 322)." A vicious circle of increasing drive with attendant stimulus generalization and more drive elevating stimuli brought above threshold evoke arousal responses. Each increase in generalization and number of suprathreshold stimuli is followed by another increment in drive, and the cycle continues repeatedly until the individual reaches some physiological limit. At this point, thought sequences are disrupted. Any stimulus-thought elicits usually remote associates which have become suprathreshold as the result of the drive increment. Thought disorder is evidenced in irrational relationships between cognitive elements, in clang association, and at its height, in word salad (i.e., the loss of appropriate grammatical connections and order in the verbalizations).

Mednick described the transition from this state to chronicity. Certain remote associates bring about a drive decrement, because they are not so closely problem oriented. The drive decrement is reinforcing, and soon much of the schizophrenic's thought may be occupied with these initially tangential associations. In the chronic phase of the disorder, drive decrement may continue until the individual

is under-aroused.

Several aspects of Mednick's theory are somewhat confusing (Epstein and Coleman, 1970). Mednick's use of the Hullian concept of drive was often equivocal. Drive is an intervening variable anchored in need deprivation and stimulus intensity and expressed in responses which are subject to measures of performance. However, Mednick often equated it to anxiety which is an avoidance motive. He further assumed that drive or anxiety can be measured directly by indexes of physiological arousal.

He, thus, confused generalized arousal with specific arousal with a definite directional component. His idea that physiological measures provide techniques for assaying anxiety further attests his confusion of the concepts and the nature of their inter-relation. Today of course, the lack of agreement between the various physiological measures in various systems under different conditions is recognized.

Mednick's use of the threshold concept also seems to be questionable. Only at that point where the dominant response is barely suprathreshold and the competing response is barely subthreshold should response competition in high-drive individuals be a special problem. Once both responses are suprathreshold, increasing drive should increase the probability of the dominant response being elicited. A concept such as response ceiling would appear to be more efficacious in the suprathreshold situation (Broen

and Storms, 1966; Storms and Broen, 1969).

Despite the difficulties cited, Mednick's theory provides a meaningful approach to the understanding of cognitive functioning in schizophrenia, particularly of verbal learning phenomena, and is deserving of further empirical testing.

> The hypotheses generated from the theory are open to direct experimental verification, and point the way to important experiments on the relationship of drive level to performance in schizophrenics and normals, and on response interference in simple and complex tasks. No matter what the outcome of such research, it should provide information of some importance (Epstein and Coleman, 1970, p. 115).

A series of studies have appeared in the research literature using verbal learning paradigms to investigate these relationships. Verbal learning tasks with little to no associative interference have been used as simple tasks, and those high in associative interference have been used as complex tasks.

Mednick and deVito (in Mednick, 1958) did not find evidence that schizophrenics learn faster than normals on low interference lists but did find their performance to be inferior to normals in the high response competition condition.

Carson (1958) investigated normals, schizophrenics, and organics using a verbal learning task. He used three experimental serial lists which differed in amount of intra-list similarity based on extra-experimental criteria. He found normals to be most affected by increased

associative interference with organics and schizophrenics being relatively unaffected. There was no significant difference between the neuropsychiatric groups.

Another study (Donahoe, Curtin, and Lipton, 1961) tested the hypothesis using serial lists. Interference was manipulated by the interpolation of similar or dissimilar material between the acquisition of a list and its relearning. The results indicated that schizophrenics and normals were equally and negatively affected by the interpolation of similar as compared to dissimilar material.

Spence and Lair (1964) used a paired-associates learning task to test Mednick's hypothesis. The experimental list in this study was composed of meaningful word pairs of low associative strength. However, a response word of high associative value for the stimulus word appeared in the list as the response term in another word pair. Thus, there was low association within word pairs but high association across pairs. The results of this study indicated that both schizophrenics and normals were adversely affected by this type of interference.

However, there was a trend in the data (nonsignificant) indicating that while schizophrenic Ss performed more poorly than normal Ss on a no-interference control list, they performed better than normal Ss on the experimental list. This trend is opposite to what would be predicted by Mednick's hypothesis and suggests that the extra-experimental manipulation of interference (i.e., based on college

student word association norms) used in this study was not as effective for schizophrenics as it might have been.

The results of this study were further confounded by several S variables. The use of nonpsychiatric hospitalized individuals as control Ss is open to question. Clinical experience suggests that such patients are susceptible to personality disruptions, sometimes of psychotic proportions, not only in the acute stage of their illness but also at various phases of convalescence (the status of the Ss in this study). This impression is strengthened by research results showing that status as a VA hospital patient (nonpsychiatric) makes individuals as susceptible to the disruptive effects of censure as are schizophrenics (Gladis and Wischner, 1962).

The actual chronicity of the schizophrenic Ss in the Spence and Lair study is also of some concern. Although current hospitalization was limited to 12 months, there was no control for number of previous admissions.

An important methodological advance in the study of associative interference in schizophrenia occurred with an investigation by Kausler, Lair, and Matsumoto (1964). They manipulated interference experimentally rather than by extra-experimental word-association norms as earlier studies had done. A mixed list with three transfer paradigms (A-C, A-Br, and C-D) was used (Twedt and Underwood, 1959). This technique assured that both normal and schizophrenic Ss were being subjected to interference.

The results of the study indicated that schizophrenic Ss made significantly more errors in the highest interference paradigm (A-Br) than did normal Ss. This result was independent of a significant main effect of groups in both List 1 and List 2 showing the schizophrenic Ss to be inferior in learning ability across lists and across paradigms. The latter finding was probably a function of the sample used in this study and is not representative of schizophrenic individuals in general.

Several factors confound the results of Kausler, et al. First, fewer schizophrenic than normal Ss reached the criterion of one perfect trial on List 1; therefore, the pre-measurement strength of S-R associations probably differed between groups. An attempt was made to check the possible effects of this inequity by doing a separate analysis using only those Ss from each group who reached the criterion of one perfect trial on List 1. The results of this analysis paralleled those of the main analysis. However, the dependent variable in the latter analysis was a questionable difference score (A-Br absolute transfer score minus A-C absolute transfer score). This particular manipulation appears to have been based on the assumption that A-Br and A-C are only quantitatively different interference paradigms. Recent evidence, however, indicates that they are also qualitatively quite different. A-C interference results from failure to inhibit List 1 response terms while A-Br interference results not only from response competition

but also from disruption of specific associations (Barnes and Underwood, 1959; Postman and Stark, 1969).

Second, the use of all chronic (lengthy hospitalization) schizophrenic Ss (there is no indication of how they were selected) confounds the interpretation of the results with institutionalization. Finally, the use of a 4:4-second exposure rate with a 10 second intertrial interval, although intended to facilitate learning in the schizophrenic Ss, may have actually been detrimental to their performance. Schizophrenic Ss often show difficulty in maintaining attention to external stimulation.

Schooler and Tecce (1967) used a mixed list with paired-associates varying in associative strength and intra-list response competition. Association values were established extra-experimentally. They tested partially remitted schizophrenics, regressed schizophrenics, and normals under conditions of positive evaluation, negative evaluation, and no evaluation. The results of this study appear to be basically uninterpretable because the measure used (change scores reflecting differences in performance from List 1 to List 2) so confounds the normative manipulation within each list with the interaction of lists that the effects cannot be differentiated.

Another study (Kapche, 1969) failed to support the interference hypothesis. However, manipulation of interference, again, was based on extra-experimental criteria. Normal and schizophrenic Ss took more trials to learn a

paired-associates list and gave fewer correct responses as interference increased. However, there was a trend in the data indicating that overt errors of all types increased for normal Ss and decreased for schizophrenic Ss as interference increased. This trend, like that noted in the Spence and Lair study, was in the opposite direction to that predicted by Mednick's theory. Once again, differential effectiveness of college student norms for the manipulation of interference in the two S groups appears to be a plausible explanation.

Streiner (1969) used a multiple choice paired-associates task to investigate interference in process and reactive schizophrenics and normals. Interference was manipulated by extra-experimental norms. He found that performance decreased across groups with increasing interference. The most salient difficulty in this investigation was the confounding of the findings by a reduction in exposure time from the low complexity task (15 seconds) to the high complexity task (8 seconds) which may have masked interference effects.

Another study attempted to replicate the Spence and Lair findings (Gonen, 1970). Schizophrenic Ss in this investigation were differentiated according to premorbid adjustment and paranoid-nonparanoid status. All schizophrenic Ss showed performance deficit on the second list, but the above S variable classifications were not significant nor did they interact with treatment. No normal

control group was used so the comparative evidence required to evaluate Mednick's hypothesis is lacking.

It is clear that the results of the above studies generally do not support an interference theory explanation of schizophrenic deficit. However, all of these studies except those of Donahoe, et al. (which yielded negative evidence) and Kausler, et al. (which yielded supporting evidence) suffer from the methodological flaw of nonexperimental manipulation of interference. It seems incongruous, and the Spence and Lair, and Kapche studies offer suggestive supporting evidence as noted, to test hypotheses about associative interference in schizophrenia using material in which the amount of interference is varied according to extra-experimental standards developed on normals. Schizophrenics, it should be recalled, are thought to suffer from disturbance of the typical pattern of association.

Furthermore, the results of those studies using high response term similarity in the second list could be predicted given the assumption about schizophrenia upon which they are based. Analysis of paired-associates learning into two functional stages (Underwood, Runquist, and Schulz, 1959; Underwood and Schulz, 1960) suggests that the high-drive schizophrenic Ss might integrate second list response terms more quickly than would normal Ss. However, their high-drive would cause them to make more errors in the associative or "hookup" stage. The net effect, then, is likely to be a performance deficit for both groups on the second list,

but no difference between the groups. The latter effect would be an artifact of the differential performances of each group in the two stages which would tend to cancel each other out across entire List 2 learning (Goulet, 1968).

Finally, all of the above studies, except that of Spence and Lair, suffer from a confounding of possible interference effects, pathology, and institutionalization. The use of chronic schizophrenic Ss in these studies does not allow the differentiation of effects attributable to the actual schizophrenic process and those which are response acretions resulting from severe and artificial circumstance (i.e., institutionalization).

The current study will attempt to improve the design to obviate the above difficulties. Interference will be manipulated experimentally and chronicity will be controlled. Further, an attempt will be made to refine the interference concept of schizophrenic deficit by dichotomizing schizophrenia into process and reactive types.

The Process - Reactive Distinction

The process-reactive distinction in schizophrenia, as the associative disturbance concept, dates from Bleuler who recognized that although some schizophrenics never recover, others do. Those who do not recover, process schizophrenics, sometimes were thought to be genetically tainted and by definition to have a poor premorbid history with an insidious onset of psychosis. Those with a better prognosis, reactive schizophrenics, are often thought to be free of

genetic taint (psychogenic in etiology) and by definition to have a good premorbid life history with a sudden onset of psychosis. Phillips (1953) developed a scale to distinguish the groups.

Several reviews (Herron, 1962; Higgins, 1964, 1969; Higgins and Peterson, 1966) indicate that the process-reactive distinction is a viable concept with an ever-growing literature. Higgins (1969) did not report any work relating interference theory and the process-reactive dimension. Work in associative processes in this area has been concerned with associative commonality.

Several relevant studies have appeared in the literature since the Higgins review. Jongsma, Sullivan, and Martin (1969) failed to find differences between process and reactive schizophrenic Ss and normal Ss in complex task conditionability. The task was learning the order of illumination of a series of lights. Their sample was small, and the criterion for their acute schizophrenic Ss allowed for relatively long periods of hospitalization (i.e., up to three years).

Irwin and Renner (1969) used simple and complex memory tasks to investigate process-reactive differences in learning under conditions of praise, censure, and non-evaluation. Their results are not supportive of an interference conceptualization, but it should be noted that the results are confounded with length of hospitalization. Also the complexity of the experimental tasks was such as to

becloud what factor or factors might have been operative in effecting the results.

The two studies cited earlier (Gonen, 1970; Streiner, 1969) which used verbal learning methodology to investigate associative interference and premorbid adjustment in schizophrenia are directly relevant. As noted both produced negative results but were open to the criticisms associated with uncontrolled length of hospitalization and the use of non-experimental means of interference manipulation.

Although the process-reactive distinction in schizophrenia has produced significant classificatory refinement and consequent increases in understandable variance in this psychopathological group with respect to many dependent variables, this has not so far been the case in associative interference. However, that the number of studies is small and their results questionable is evident.

Verbal Learning Methodology

In addition to the methodological improvements of experimental manipulation of associative interference and of dichotomizing the schizophrenic Ss along the process-reactive continuum, the present study will further control interference by using an overlearning technique (Postman, 1962a). This approach plus the adjunct of collecting modified, modified free recall (MMFR) data is not only concerned with interference but also with the "fate" of the first-list associations in the various transfer paradigms (Barnes and Underwood, 1959). Analysis of intrusions may provide

additional relevant information.

Barnes and Underwood (1959), using a normal population, concluded that first-list associations are extinguished in the A-B, A-C (old stimulus-new response) paradigm. Postman (1962b), again using a normal population, but with a mixed list approach obtained results which are highly comparable with those of Barnes and Underwood using homogeneous lists. Other studies have consistently confirmed the inhibition or extinction of first list response terms in the A-C paradigm (Delprato and Garskof, 1969; Postman, 1962a; Postman and Stark, 1969; Solso, 1969).

In the A-B, A-Br (old stimulus-response from original list but previously paired with another stimulus) paradigm, several studies have found a high correlation between losses in List 1 MMFR and negative transfer (Postman, 1962a; Solso, 1969). Postman and Stark (1969) used five transfer groups and two conditions of practice (Recall method and Multiple-choice method) to investigate the effect of response availability in transfer and interference. The paradigms tested were: C-D, C-B, A-C, A-Br, A-B'. Recall and test trials were alternated under each condition. List 1 recall data was collected at the end of second list learning.

In condition Multiple-choice, Postman and Stark found that A-Br was the only paradigm to show appreciable retroactive inhibition (RI). The classical paradigm of unlearning, A-C, showed only 5% RI. In condition Recall, all paradigms showed considerable RI; it was greatest for A-C,

followed in order by A-Br, A-B', C-B, and C-D. All paradigms showed greater RI in condition Recall than in condition Multiple-choice. This difference was greatest for A-C and smallest for C-B with A-Br falling between. The investigators concluded,

> In general, the increases are greater (a) when the responses in the two lists are different than when they are the same, and (b) when the stimuli remain the same than when they change (p. 173).

Further analysis indicated that A-Br was the only paradigm showing significantly more RI than a rest group in condition Multiple-choice. In condition Recall, reliable RI was found for the A-C, A-Br, and A-B' paradigms when compared to an appropriate rest group.

Analysis for interference with specific associations (i.e., where responses may be assumed to be fully available but the bond between them disturbed or lost) indicated that A-Br was the only paradigm showing such an effect.

The above findings are important to the present study in that they confirm that the interference found in the A-C paradigm probably results from response competition, and that successful learning of List 2 pairs depends on the inhibition of List 1 response terms. Disruption or extinction of specific associations does not occur in this paradigm. However, in the A-Br paradigm, loss of specific associations (true associative interference) does occur in addition to response competition. Thus, although A-C and A-Br are roughly "interference paradigms," there appears to be a difference in the nature of their mechanisms.

What, then, are the effects of overlearning or overtraining on List 1 for paradigms A-C and A-Br? Postman (1962a) investigated the effect of overlearning on several transfer paradigms including A-C and A-Br. He used meaningful words. His data reflected a decrease in A-C negative transfer (as measured by mean number of correct responses in the first 10 trials of List 2) with increased first list practice. The A-Br paradigm, however, showed steadily increasing negative transfer with overlearning. The List 1 MMFR data also reflected these differences. In the low overlearning condition A-Br responses were recalled better than A-C responses, but with increasing number of List 1 reinforcements the paradigms switched positions so that in the high overlearning condition A-C responses were recalled better than A-Br responses.

Solso (1969) used high and low meaningful trigrams (CVCs) to investigate the effects of massive overlearning (100%) on A-C, A-Br, and C-D transfer paradigms in a mixed list. He found reduced negative transfer in the A-Br paradigm with overlearning. This is contradictory to Postman's finding with meaningful words and Jung's (1962) earlier finding with CVCs.

A series of studies by James and Greeno (1970) confirmed the Postman and the Jung findings with adjectives except where the list was relatively short (six pairs). With short lists or with digits (usually considered meaningful material) overlearning had no effect.

The majority of the studies dealing with the effects of overlearning on transfer of training support the hypothesis that with meaningful words and normal Ss overlearning increases interference in the A-Br paradigm. Furthermore, overlearning may decrease the amount of interference in the A-C paradigm, although it did not result in positive transfer in any of the studies reviewed here. This has occured in some studies (see Mandler, 1962). Finally, Postman's study suggests that complementary findings will appear in List 1 MMFR data for each paradigm. No one has investigated the effects of overlearning on transfer in a schizophrenic group.

One final methodological issue is of concern. Twedt and Underwood (1959) demonstrated that transfer effects were essentially comparable for mixed and unmixed lists in all of the paradigms they tested (these included those used in the present study: A-Br, A-C, and C-D). Since that time some investigators have found contradictory results. Slamecka (1967) for example found negative transfer for the A-Br paradigm in a mixed list but positive transfer for the same paradigm in an unmixed list. The question of the comparability of results employing the different procedures appears to be a methodological problem which has not yet been satisfactorily answered. The present study, however, will employ a mixed list design in keeping with the earlier work of Kausler et al. (1964). This approach seems justifiable in light of the reduction in experimental session length made

possible by such an approach, which is important with schizophrenic Ss, and in light of the major thrust of the study which concerns the performance of a pathological group on a task in which associative interference is present.

The Problem

Mednick's theory makes differential predictions about the drive level of acute and chronic schizophrenics. The former are thought to be in a state of over-arousal, and the latter are thought to be under-aroused. The hypothesis that Mednick put forth concerning associative interference in schizophrenia depends on the assumption that high drive results in response competition. However, the verbal learning studies intended to elucidate this phenomenon, with the single exception of Spence and Lair (1964), have used chronic schizophrenic Ss. Furthermore, the use of chronic schizophrenic patients confounds any findings with the extrinsic effects of institutionalization.

It seemed that this undesirable consequence could be avoided by classifying schizophrenics as process or reactive (a measure of their pre-hospital history of disorder) and by keeping hospitalization to a minimum. This notion gained further support from the existing literature which suggested (as did clinical experience) that process and reactive schizophrenics might differ in drive level in a manner similar to that suggested by Mednick for chronic and acute schizophrenics.

Several reviews of the process-reactive literature (Fowles, Watt, Maher, and Grinspoon, 1970; Higgins, 1969)

indicate that studies of arousal using physiological measures have resulted in confused and often conflicting findings. The principle emerging from these studies is that there is little direct covariance among physiologic systems and measures. On a behavioral level, however, many studies have found process schizophrenics to respond with minimal energy and reactives with much energy in a variety of tasks (Crider, Grinspoon, and Maher, 1965; Donoghue, 1964; Higgins, Mednick, Philip, and Thompson, 1966; Higgins, Mednick, and Thompson, 1966; Reisman, 1960; Reynolds, 1965; Smith, 1961; Zlotowski and Bakan, 1963). Only two studies offered nonsupportive results (Klein, Cicchette, and Spohn, 1967; Schweid, 1966). With respect to behavioral reactivity, then, good and poor premorbid schizophrenics may be thought to fall at the opposite ends of an inverted-U curve representing the relationship between drive and performance. Both groups might be expected to perform more poorly than normals but for different reasons; poor premorbids because of too little arousal or drive and good premorbids because of too much reactivity. Task complexity (i.e., degree of associative interference) would be expected to interact with reactivity to affect performance.

At a behavioral level, the following prediction was made: That the performance of reactive schizophrenics would be more negatively affected by increased associative interference in a task than would the performance of process schizophrenics or normals.

Method

Subjects

The Ss were 60 hospitalized, male schizophrenic patients and 30 male hospital employees. The schizophrenic Ss were obtained through the Central Testing Service of a large VA Hospital in which all first and second admissions are routinely examined. They were classified as either good or poor premorbid schizophrenia using a questionnaire self-report scored on a revision of the Phillips Scale of Premorbid Adjustment in Schizophrenia (Phillips, 1953; DeWolfe, 1968). Those scoring 12 or below on the scale were considered to be good premorbids and those scoring 18 or above were considered to be poor premorbids. Within the limitations imposed by matching Ss were taken consecutively until there were 30 Ss in each category. The diagnosis of schizophrenia was made according to the usual neuropsychiatric criteria. Any patient who had ever carried a diagnosis of organic brain damage, alcoholism, or drug addiction was excluded. The schizophrenic sample was further restricted by using only acute patients, i.e., those with less than one year current hospitalization.

The male hospital employee Ss were volunteers obtained through various hospital departments, i.e., Supply, Engineering, Nursing, etc. These normal Ss were given a

non-psychiatric equivalent of the Phillips scale, and only those scoring 12 or below (i.e., "good premorbid") were used in the study.

Lists

A mixed list paired-associates learning task (Twedt and Underwood, 1959) was used. Three transfer paradigms were represented: A-B, A-C (old stimulus-new response) A-B, A-Br (old stimulus-response from original list but previously paired with another stimulus), and A-B, C-D (new stimulus and new response). Lists 1 and 2 each contained six pairs of words from the Russell and Jenkins (1954) revision of the Kent Rosanoff Word Association Test. No stimulus or response word was an associate of any other word with a frequency greater than 1/1008. All words comprising the list were roughly equated for meaningfulness as indicated by familiarity using the Lorge-Thorndike (1944) word count. The words had a frequency of appearance greater than 37 per million. All words began with a different letter and were screened for formal similarities (i.e., rhymes, or logical categories). Thirteen of the eighteen words were nouns and the rest were adjectives. Pairs were established using a random procedure with the exception that no adjective appeared as a stimulus..

List 2 contained two pairs for each of the three paradigms and was identical for all Ss. List 1 was used with three variations allowing each word pair to be employed in each of the three paradigms as described by Twedt and

Underwood (1959) and Kausler and Kanoti (1963). Thus confounding of paradigmatic effects with specific properties of the word pairs (e.g., differential acquisition rates) was controlled. Four different random arrangements of each List 1 paradigmatic variation and of List 2 were employed to eliminate possible serial order effects. The experimental lists appear in the Appendix.

Procedure

Each S was given a practice list (PL) of three word pairs which were presented with modified paired-associates learning instructions. This list (with four serial order variations) was learned by the anticipation method to a criterion of 15 trials or 1 perfect recitation, whichever occurred first. List 1 was learned to a criterion of one perfect recitation. Half of the Ss in each group were then given additional trials to a criterion of 50% overlearning. The schizophrenic Ss classified according to premorbid adjustment and overlearning (OL) or no overlearning (N) were matched for initial learning rate as indicated by the number of trials involved in learning List 1 to a criterion of one perfect recitation. Although the normal Ss were not matched to the schizophrenic Ss for initial learning rate, they were not found to be significantly different from the pathological groups with respect to first list acquisition. Relevant means and standard deviations appear in Table 1. The analysis of variance appears in Table 2. There were no significant differences between the S groups in age or

Table 1

Means and Standard Deviations
for Schizophrenic List 1 Acquisition
Classified for 2 Degrees of Learning
(OL=overlearning, N=no overlearning)

Group*	Number of Trials to Criterion	
	Mean	S.D.
Process-OL	15.67	8.09
Reactive-OL	16.27	10.87
Normal-OL	11.20	7.19
Process-N	14.07	8.78
Reactive-N	13.33	6.24
Normal-N	12.53	6.24

*N=15 in each Group

Table 2

Analysis of Variance

for Schizophrenic and

Normal List 1 Acquisition

Source	df	MS	F	P
Groups	5	54.63	<1.00	NS
Error	84	69.91		
Total	89			

education. Relevant data and analyses appear in Tables 3 and 4.

A two minute period of informal conversation intervened between the completion of List 1 learning and the beginning of practice on List 2. List 2 learning continued to a criterion of 1 perfect recitation or 10 trials, whichever occured first. A 2:2-second exposure rate and a 4-second intertrial interval were employed with all lists. Material was presented to the Ss with a 303-C Layfayette Memory Drum. All of the Ss' responses were recorded to allow for an analysis of errors.

Immediately following the completion of List 2 learning, modified, modified free recall (MMFR) data was collected (Barnes and Underwood, 1959; Briggs, 1954; Melton, 1961). The Ss were provided with a list of the stimulus words for this experiment. Each word was followed by the appropriate number of blanks (i.e., one or two). The Ss were asked to fill in the response terms as they came to mind. Two minutes were allowed for this initial recall. The Ss were then asked to designate by writing a 1 or a 2 next to each recalled response the list from which each response came. An additional two minutes were allowed for this.

Analyses

In the main analysis List 2 errors were used as the dependent variable in a 3x2x3 analysis of variance (groups x degree of original learning x paradigms), where paradigms

Table 3

Means and Standard Deviations

for Age and Education

Group*	Age		Education	
	Mean	S.D.	Mean	S.D.
Process	37.27	9.94	11.93	2.13
Reactive	35.50	10.80	12.27	2.09
Normal	37.00	10.24	12.87	2.29

*N=30 in each Group

Table 4

Analyses of Variance

for Age and Education

Source	df	MS	F	P
Age	2	27.21	<1.00	NS
Error	87	110.52		
Total	89			
Education	2	6.71	1.28	NS
Error	87	5.26		
Total	89			

...d. The difference score represents loss of specific associations.

Finally, specific List 1 response intrusions into List 2 A-C paradigm were analyzed in a 3x2 analysis of variance (groups x degree of original learning).

Results

Transfer

Analysis of variance on List 2 errors showed significant main effects of interference (A-Br paradigm plus A-C paradigm vs. C-D paradigm) and type of interference (A-Br paradigm vs. A-C paradigm). The p values were $p < .01$ in both cases. These findings indicate that the experimental manipulation of associative interference was successful when viewed across groups. As expected more errors occured in the A-Br paradigm than in the A-C paradigm. The C-D paradigm showed the smallest number of errors.

A main effect of pathology was also found ($p < .05$). Thus, as a group the schizophrenic Ss made more errors on List 2 than did the normal Ss. A significant interaction ($p < .05$) was found between type of interference (A-C paradigm or A-Br paradigm) and type of schizophrenia (process or reactive). Process schizophrenic Ss made an equal number of errors in both paradigms, while reactive Ss made fewer errors in the A-C paradigm than in A-Br. The reactives' performance, thus, paralleled that of the normal Ss. The means and standard deviations for the above results appear in Table 5. The analysis of variance appears in Table 6. There were no significant differences between the groups in the control paradigm (C-D). Therefore, a separate

Table 6

Analysis of Variance of List 2 Errors

Source	df	MS	F	P
Pathology (P)	1	274.49	5.51	<.05
Type of Schizophrenia (TS)	1	2.00	<1.00	NS
Degree of Original Learning (DOL)	1	38.53	<1.00	NS
DOL x P	1	16.02	<1.00	NS
DOL x TS	1	92.45	1.86	NS
SS/Groups	84	49.78		
Interference (I)	1	716.45	52.03	<.01
I x P	1	42.40	3.08	NS
I x TS	1	18.05	1.31	NS
I x DOL	1	0.27	<1.00	NS
I x DOL x P	1	0.83	<1.00	NS
I x DOL x TS	1	10.41	<1.00	NS
I x SS/Groups	84	13.77		
Type of Interference (TI)	1	112.02	14.64	<.01
TI x P	1	5.39	<1.00	NS
TI x TS	1	32.03	4.19	<.05
TI x DOL	1	8.89	1.16	NS
TI x DOL x P	1	2.17	<1.00	NS
TI x DOL x TS	1	14.71	1.92	NS
TI x SS/Groups	84	7.65		
Total	89			

analysis using absolute transfer scores (C-D errors minus experimental paradigm errors) was deemed unnecessary since the results would parallel the raw score analysis. The graphical presentation of List 2 error data in terms of absolute transfer, however, follows convention and is presented in Figure 1.

MMFR

Analysis of variance on List 1 recall scored under lenient criteria (i.e., response recall was scored irrespective of proper list membership designation) revealed main effects of both interference ($p < .01$) and type of interference ($p < .01$). Once again, the experimental manipulation of interference was successful. All paradigms were found to be significantly different from each other, and the differences were in the expected directions. Recall was best in the control paradigm (C-D), less in the A-Br paradigm and worst in the A-C paradigm.

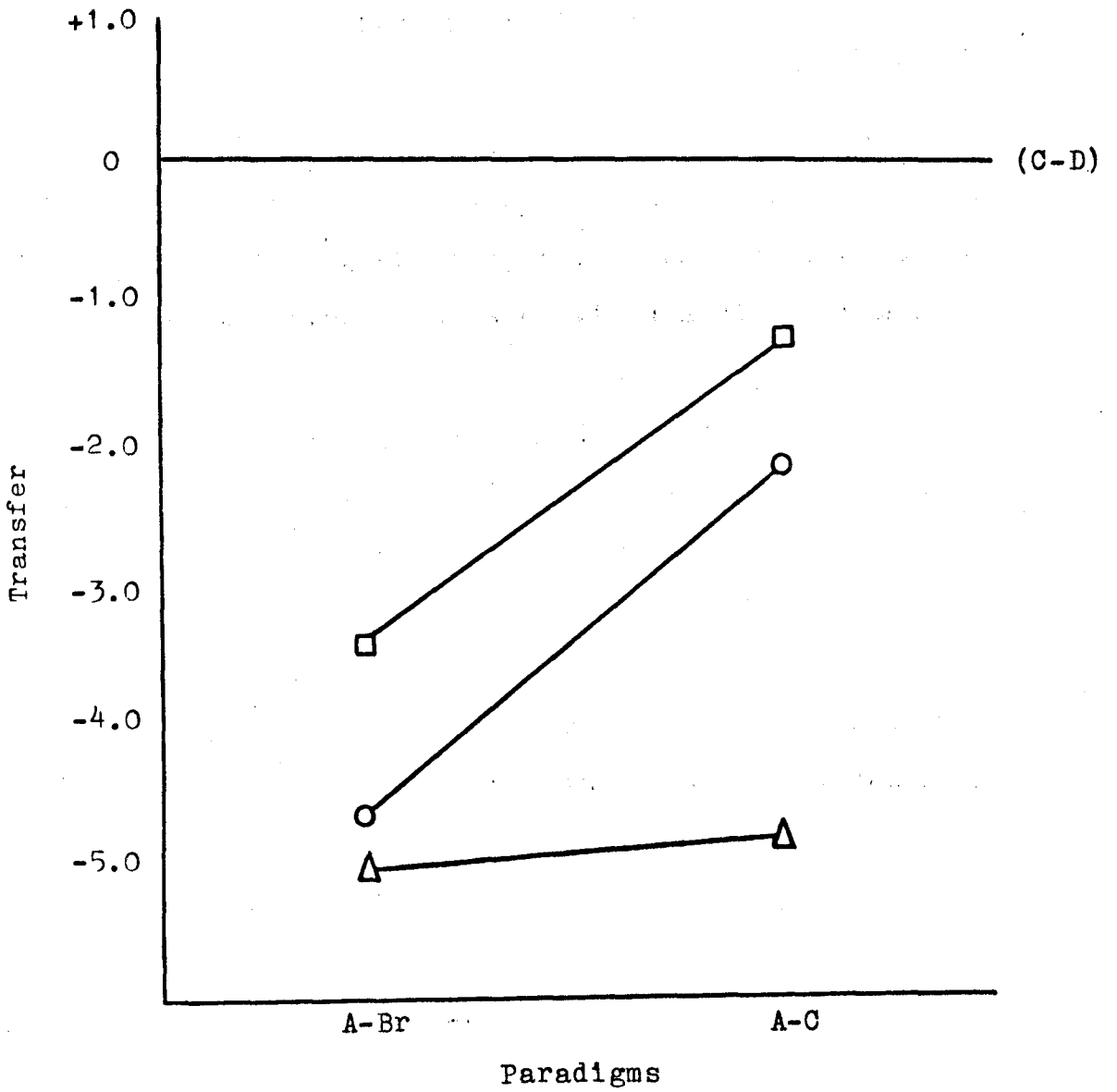
A significant interaction of interference and type of schizophrenia was found ($p < .05$). Process schizophrenic Ss recalled List 1 responses in the interference paradigms (A-Br plus A-C) better than did reactive schizophrenic Ss. The schizophrenic groups, however, did not differ greatly in recall in the control paradigm.

Finally, a significant triple interaction (type of interference x degree of original learning x type of schizophrenia) was found ($p < .05$). In the overlearning condition process schizophrenic Ss recalled List 1 responses in the

Figure 1

Amount of Negative Transfer

in Mean Number of Errors on List 2



A-Br paradigm to a greater extent than did reactive Ss, and this difference was significantly greater than that found in the A-C paradigm. Without overlearning, process Ss recalled more List 1 responses in the A-C paradigm than did reactive Ss; no difference between the groups was found in the A-Br paradigm. The above data are presented in Table 7. The analysis of variance is presented in Table 8, and a graphical presentation of the data appears in Figure 2.

List 1 MMFR data were also scored using strict criteria (i.e., requiring correct list membership designation in addition to correct recall). Subtraction of strict scores from lenient scores provided a difference score which was an indicator of loss of specific associations. Others (e.g., Postman and Stark, 1969) have used mean differences between lenient and strict scoring to compare groups for such losses. Analysis of variance on these difference scores revealed a main effect of type of interference ($p < .01$). All other terms in this analysis were non-significant. These findings reflect losses of specific associations, across groups, in the A-Br and C-D paradigms which are significantly greater than losses in the A-C paradigm. The data and analysis appear in Tables 9 and 10.

Analysis of variance was also carried out on List 2 MMFR data (presented in Tables 11 and 12) with lenient scoring criteria. The main effects of interference and of type of interference were found to be significant ($p < .01$ and $p < .05$, respectively). Thus, the differences between paradigms were significant; recall in paradigm C-D was

Table 7

Means and Standard Deviations

for List 1 MMFR (lenient scoring)

Classified for 2 Degrees of Original Learning

(OL=overlearning, N=no overlearning)

Group*	Paradigm					
	A-Br		A-C		C-D	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Process-OL	1.46	0.64	1.14	0.60	1.40	0.62
Reactive-OL	1.06	0.79	0.94	0.68	1.80	0.40
Normal-OL	1.20	0.84	1.06	0.79	1.66	0.62
Process-N	1.00	0.81	1.06	0.69	1.20	0.81
Reactive-N	1.06	0.78	0.46	0.62	1.14	0.71
Normal-N	1.46	0.63	1.06	0.78	1.46	0.63

*N=15 in each Group

Table 8

Analysis of Variance of

List 1 MMFR (lenient scoring)

Source	df	MS	F	P
Pathology (P)	1	1.90	2.29	NS
Type of Schizophrenia (TS)	1	0.80	<1.00	NS
Degree of Original Learning (DOL)	1	2.69	3.24	NS
DOL x P	1	1.67	2.01	NS
DOL x TS	1	0.20	<1.00	NS
SS/Groups	84	0.83		
Interference (I)	1	7.83	20.83	<.01
I x P	1	0.01	<1.00	NS
I x TS	1	2.01	5.15	<.05
I x DOL	1	0.81	2.08	NS
I x DOL x P	1	0.07	<1.00	NS
I x DOL x TS	1	0.65	1.67	NS
I x SS/Groups	84	0.39		
Type of Interference (TI)	1	2.94	9.80	<.01
TI x P	1	0.00	<1.00	NS
TI x TS	1	0.42	1.40	NS
TI x DOL	1	0.14	<1.00	NS
TI x DOL x P	1	0.13	<1.00	NS
TI x DOL x TS	1	1.40	4.67	<.05
TI x SS/Groups	84	0.30		
Total	89			

Figure 2

Mean Number of Responses Recalled in List 1 MMFR Classified for 2 Degrees of Original Learning (OL=overlearning, N=no overlearning)

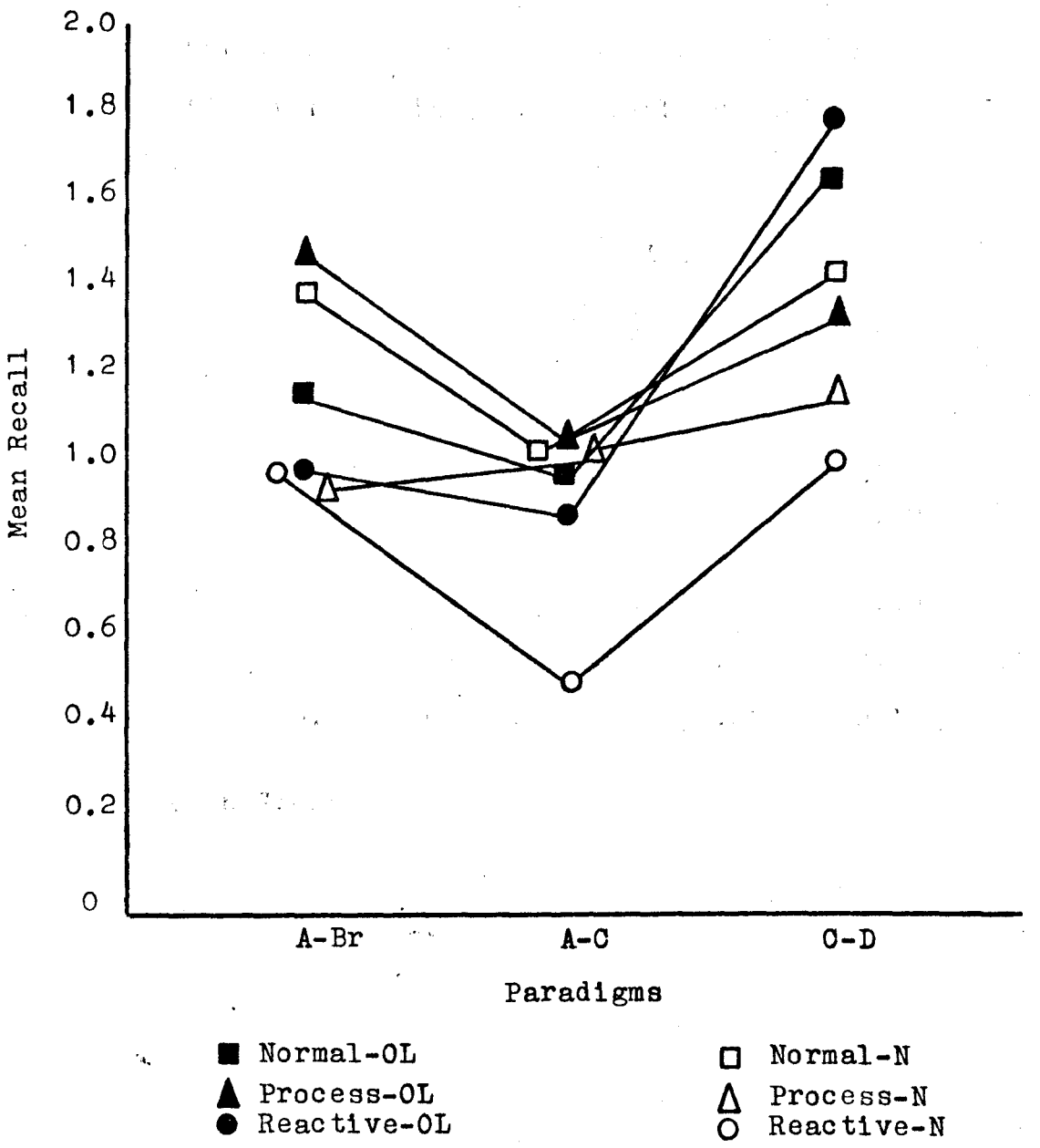


Table 9

Means and Standard Deviations

for List 1 MMFR Difference Scores

Classified for 2 Degrees of Original Learning

(OL=overlearning, N=no overlearning)

Group*	Paradigm					
	A-Br		A-C		C-D	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Process-OL	0.20	0.54	0.00	0.00	0.27	0.45
Reactive-OL	0.20	0.54	0.13	0.22	0.40	0.61
Normal-OL	0.00	0.00	0.00	0.00	0.20	0.54
Process-N	0.40	0.61	0.13	0.33	0.20	0.40
Reactive-N	0.13	0.45	0.00	0.00	0.20	0.40
Normal-N	0.40	0.49	0.07	0.26	0.13	0.33

*N=15 in each Group

Table 10

Analysis of Variance for

List 1 MMFR Difference Scores

Pathology (P)	1	0.15	<1.00	NS
Type of Schizophrenia (TS)	1	0.05	<1.00	NS
Degree of Original Learning (DOL)	1	0.09	<1.00	NS
DOL x P	1	0.31	1.82	NS
DOL x TS	1	0.45	2.65	NS
SS/Groups	84	0.17		
Interference (I)	1	0.60	3.16	NS
I x P	1	0.07	<1.00	NS
I x TS	1	0.22	1.16	NS
I x DOL	1	0.75	3.95	NS
I x DOL x P	1	0.05	<1.00	NS
I x DOL x TS	1	0.04	<1.00	NS
I x SS/Groups	84	0.19		
Type of Interference (TI)	1	1.42	17.75	<01
TI x P	1	0.00	<1.00	NS
TI x TS	1	0.08	1.00	NS
TI x DOL	1	0.21	2.62	NS
TI x DOL x P	1	0.24	3.00	NS
TI x DOL x TS	1	0.01	<1.00	NS
TI x SS/Groups	84	0.08		
Total	89			

Table 11

Means and Standard Deviations

for List 2 MMFR (lenient scoring)

Classified for 2 Degrees of Original Learning

(OL=overlearning, N=no overlearning)

Group*	Paradigm					
	A-Br		A-C		C-D	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Process-OL	1.40	0.80	1.60	0.61	1.80	0.40
Reactive-OL	1.33	0.79	1.40	0.88	1.67	0.58
Normal-OL	1.67	0.69	1.73	0.46	2.00	0.00
Process-N	1.33	0.60	1.20	0.83	1.87	0.48
Reactive-N	1.13	0.81	1.80	0.52	1.80	0.40
Normal-N	1.33	0.79	1.53	0.62	1.87	0.32

*N=15 in each Group

Table 12

Analysis of Variance of

List 2 MMFR (lenient scoring)

Source	df	MS	F	P
Pathology (P)	1	1.56	2.33	NS
Type of Schizophrenia (TS)	1	0.00	<1.00	NS
Degree of Original Learning (DOL)	1	0.45	<1.00	NS
DOL x P	1	0.66	<1.00	NS
DOL x TS	1	0.67	1.00	NS
SS/Groups	84	0.67		
Interference (I)	1	8.57	30.61	<.01
I x P	1	0.00	<1.00	NS
I x TS	1	0.18	<1.00	NS
I x DOL	1	0.36	1.28	NS
I x DOL x P	1	0.02	<1.00	NS
I x DOL x TS	1	0.17	<1.00	NS
I x SS/Groups	84	0.28		
Type of Interference (TI)	1	1.42	4.30	<.05
TI x P	1	0.04	<1.00	NS
TI x TS	1	0.84	2.55	NS
TI x DOL	1	0.23	<1.00	NS
TI x DOL x P	1	0.01	<1.00	NS
TI x DOL x TS	1	1.62	4.91	<.05
TI x SS/Groups	84	0.33		
Total	89			

greater than in paradigm A-C which was in turn greater than in A-Br. A significant triple interaction (type of interference x degree of original learning x type of schizophrenia) was also present. With overlearning, process schizophrenic Ss recalled a greater number of List 2 responses in the A-C paradigm than did reactives. There was little difference between the two groups in the A-Br paradigm. However, without overlearning, reactive Ss recalled more List 2 response terms than did process Ss in the A-C paradigm. This difference was significantly greater than the difference between the groups in the A-Br paradigm.

Analysis of List 2 MMFR difference scores (lenient scoring minus strict scoring) showed a main effect of type of interference ($p < .01$) and two significant triple interactions (a) type of interference x degree of original learning x pathology and, b) type of interference x degree of original learning x type of schizophrenia). The p values were $p < .05$ for both interactions. The relevant data and analysis appear in Tables 13 and 14. Across groups, the A-Br and C-D paradigms were associated with significantly greater losses in specific associations than was the A-C paradigm. With overlearning, the schizophrenic Ss showed more loss of specific associations in the A-Br paradigm than did normal Ss, and the difference between the two groups was significantly greater than in the A-C paradigm. Conversely, without overlearning, the schizophrenic Ss showed more loss of specific associations in the A-C

Table 13

Means and Standard Deviations

for List 2 MMFR Difference Scores

Classified for 2 Degrees of Original Learning

(OL=overlearning, N=no overlearning)

Group*	Paradigm					
	A-Br		A-C		C-D	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Process-OL	0.07	0.26	0.00	0.00	0.07	0.26
Reactive-OL	0.33	0.47	0.13	0.35	0.00	0.00
Normal-OL	0.00	0.00	0.00	0.00	0.07	0.26
Process-N	0.20	0.40	0.07	0.26	0.40	0.71
Reactive-N	0.07	0.26	0.13	0.33	0.13	0.50
Normal-N	0.20	0.40	0.00	0.00	0.13	0.50

*N=15 in each Group

Table 14

Analysis of Variance for

List 2 MMFR Difference Scores

Source	df	MS	F	P
Pathology (P)	1	0.27	1.80	NS
Type of Schizophrenia (TS)	1	0.00	<1.00	NS
Degree of Original Learning (DOL)	1	0.37	2.47	NS
DOL x P	1	0.01	<1.00	NS
DOL x TS	1	0.56	3.73	NS
SS/Groups	84	0.15		
Interference (I)	1	0.07	<1.00	NS
I x P	1	0.01	<1.00	NS
I x TS	1	0.62	2.82	NS
I x DOL	1	0.36	1.64	NS
I x DOL x P	1	0.26	1.18	NS
I x DOL x TS	1	0.00	<1.00	NS
I x SS/Groups	84	0.22		
Type of Interference (TI)	1	0.36	7.20	<.01
TI x P	1	0.00	<1.00	NS
TI x TS	1	0.01	<1.00	NS
TI x DOL	1	0.00	<1.00	NS
TI x DOL x P	1	0.24	4.80	<.05
TI x DOL x TS	1	0.27	5.40	<.05
TI x SS/Groups	84	0.05		
Total	89			

paradigm than did normal Ss, and the difference between the two groups was significantly greater than in the A-Br paradigm. Specifically, reactive schizophrenic Ss lost more specific associations in the A-Br paradigm than did process Ss, with overlearning, and the difference between the two groups was significantly greater than in the A-C paradigm. Without overlearning the converse held true. Process Ss lost more specific associations in the A-Br paradigm than did reactive Ss, and the difference between the two groups was significantly greater than in the A-C paradigm.

Intrusions

The above results suggested that an analysis of intrusion errors might provide additional information about differential group performances. Specific intrusions (List 1 responses appearing in List 2 to the appropriate stimuli) were analyzed for the A-C paradigm. Intrusions of this type were not analyzed in the A-Br paradigm, because they appeared to be approximately equal in both schizophrenic groups and because specific intrusions are completely confounded with intralist errors in this paradigm. The data and analysis appear in Tables 15 and 16, respectively.

The main effects of pathology and of type of schizophrenia were found to be significant ($p < .01$). The degree of original learning x pathology interaction was also significant ($p < .01$). The analysis indicated that all groups were significantly different from each other when considered across degrees of original learning.

Table 15

Means and Standard Deviations

for Specific Intrusions in List 2

A-C Paradigm Learning

Classified for 2 Degrees of Original Learning

(OL=overlearning, N=no overlearning)

Groups*	Mean	S.D.
Process-OL	0.54	0.71
Reactive-OL	0.34	0.82
Normal-OL	0.00	0.00
Process-N	0.34	0.69
Reactive-N	0.06	0.26
Normal-N	0.20	0.75

*N=15 in each Group

Table 16

Analysis of Variance

for Specific Intrusions in

List 2 A-C Paradigm Learning

Source	df	MS	F	P
Pathology (P)	1	0.94	13.43	$<.01$
Type of Schizophrenia (TS)	1	0.81	11.57	$<.01$
Degree of Original Learning (DOL)	1	0.18	2.57	NS
DOL x P	1	0.93	13.28	$<.01$
DOL x TS	1	0.02	<1.00	NS
SS/Groups	84	0.07		
Total	89			

Process schizophrenic Ss made more specific intrusion errors than did reactive Ss, who in turn made more errors than normal Ss. Also, schizophrenic Ss made more intrusion errors in the overlearning condition than did normal Ss, and this difference was significantly greater than it was in the no overlearning condition.

Discussion

The main methodological results of this study will be discussed first. The interactions with pathology will be more clear once this has been done. The results of this study affirm the efficacy of the verbal learning methodology used to manipulate associative interference. Of 12 main effects attributable to paradigmatic variation, 10 yielded results significant beyond the .05 level; most were significant far beyond .001. One of the two remaining effects was significant at the .10 level, and both of these results were found in MMFR difference score analyses (lenient scoring minus strict scoring). Furthermore, most of these results were in accord with expectations based on both prior findings and theory. Those that were not expected provide insight into the mechanisms of interference in the various paradigms and, for the most part, are compatible with recent findings of verbal learning researchers.

The analysis of List 2 errors (Tables 5 and 6) provided paradigmatic findings in accord with expectation. The A-Br paradigm showed more interference than A-C which in turn showed greater interference than the no interference control paradigm (C-D). These results paralleled the findings with normal populations (e.g., college students).

The findings in List 1 MMFR under lenient scoring (Tables 7 and 8) were concordant with expectation based on retroactive inhibition of List 1 response terms in the A-C paradigm. Greater A-Br than A-C recall was expected given that the same response terms were required in both List 1 and List 2 for this paradigm. That is, as appropriate List 1 responses were being inhibited during List 2 learning the same responses were being reinforced with respect to different stimuli. The probability of recall of the response, then, remained relatively high. Of course, C-D, as expected, showed the best response recall, because no experimental associative interference was present in this paradigm.

List 1 MMFR analysis for loss of specific associations (Tables 9 and 10) yielded evidence (i.e., significant main effect of type of interference) which provided some support for the immunity of the A-C paradigm to disruption due to loss of specific associations (Postman and Stark, 1969). However, appearance of these losses at an equal level in the C-D paradigm suggested that their relative absence in A-C was contingent upon failure of recall in this paradigm. Thus, analysis under lenient and strict scoring would be redundant in this respect. Yet, the occurance of loss of specific associations was higher in the C-D paradigm for this study than in that of Postman and Stark. No difference between lenient and stringent scoring was found for C-D in that study. The present

study employed a mixed list technique whereas Postman and Stark did not; the mixed list technique might have allowed generalization of disruption of specific associations across paradigms.

The results of List 2 MMFR under lenient scoring criteria (Tables 11 and 12) were somewhat surprising. The C-D paradigm was expected to show best recall followed in order by A-Br and A-C. However, superiority of A-C recall to A-Br recall was found. Many investigators have paid little attention to second list retention, but those who have (e.g., Postman, 1962a) reported uniform recall across paradigms with college student Ss.

The present findings were seen as being compatible with those of Postman and Stark (1969). It seemed likely that the disruption of specific associations in the A-Br paradigm contributed to the loss of response terms during second list learning at a rate greater than that attributable to retroactive inhibition alone. These losses appeared in List 2 recall, where, without such losses, high response availability would have been expected. List 2 difference scores (Tables 13 and 14) provided additional support. Equally high losses of specific associations appeared in paradigms A-Br and C-D with little loss in A-C. These findings parallel the paradigmatic differences noted earlier in List 1 MMFR. However, unlike the situation for List 1, where no distinction between loss of specific associations and low response availability could be made, here

one of the high recall paradigms under lenient scoring (A-C) showed low disruption of specific associations. Thus, loss of specific associations was not necessarily dependent on response availability. That this should be true for the other paradigms seemed reasonable. In this light, MMFR for both lists reflected the relative immunity of the A-C paradigm to disruption of specific associations, the expected disruption in paradigm A-Br, and the highly unexpected susceptibility of C-D to such disruption in a mixed list.

The other major verbal learning methodological technique (i.e., the use of two degrees of original learning) employed in this study did not produce any significant main effects. The expected interaction with the A-Br paradigm to create especially high associative interference (List 2 errors) was not found. Although there was no indication from the present data concerning the reason for this negative result, the relatively short list length (six pairs) seemed to provide a likely answer (James and Greeno, 1970).

However, the degree of original learning was found to interact significantly with pathology in the analysis of specific List 1 intrusions in List 2 A-C learning (Tables 15 and 16). Complex interactions between this factor and those of type of interference and type of schizophrenia in the MMFR data were also found.

The major findings of this study can now be understood in light of the particular methodology used. The main effect of pathology found in the analysis of List 2 errors

(Tables 5 and 6) resulted, at least in part, from the sensitivity of the schizophrenic Ss to the interference created in the experimental paradigms. Two factors indicated that the greater number of schizophrenic errors did not result from generally impaired learning ability, attentional deficit, or flagging motivation. First the groups were matched for initial learning ability as indicated by List 1 acquisition rate (i.e., number of trials to criterion). Second, there were no significant differences between groups in the C-D control paradigm. In general, the results of Kausler et al. were confirmed. The performance of schizophrenic individuals was more disrupted by associative interference than was the performance of normal individuals. Further, the use of experimental procedures to manipulate interference in comparison to extra-experimental or normative techniques (as done in most associative interference in schizophrenia studies) was supported.

The finding of a significant type of interference x type of schizophrenia interaction in the List 2 error data was directly related to the hypothesis upon which this study was based. From Figure 1, it was clear that the performance of reactive or good premorbid schizophrenic Ss, while impaired, paralleled the performance of normal Ss with respect to the relationship between errors in the A-Br and A-C paradigms. However, the performance of process or poor premorbid Ss differed from that of the other two groups; it did not improve in paradigm A-C as would have been expected.

Given the nature of the A-C paradigm, the most tenable explanation of this finding appeared to be that process schizophrenic Ss failed to inhibit or extinguish first list response terms to a greater extent than did the other two groups. List 1 MMFR (lenient scoring) provided some supporting evidence (Tables 7 and 8). The triple interaction (type of interference x degree of original learning x type of schizophrenia) indicated that without overlearning process Ss recalled List 1 response terms in the A-C paradigm much better than did reactive Ss. In fact poor premorbid Ss recalled A-C responses as well as A-Br responses under these conditions. This was not true of good premorbid schizophrenic Ss or normal Ss. Both of these groups tended to show a response recall decrement in the A-C paradigm relative to A-Br. However, overlearning on List 1 appeared to eradicate the recall decrement in A-C for reactive Ss and normal Ss. It also slightly increased process schizophrenic recall in paradigm A-Br. Simply, process schizophrenic Ss appeared to perseverate List 1 responses in paradigms A-C and A-Br. Overlearning appeared to increase this perseveration in the A-Br paradigm relative to reactive Ss and appeared to make the inhibition of first list response terms in paradigm A-C more difficult for reactive schizophrenic and normal Ss.

Strong support for this interpretation was found in paradigm specific intrusions in A-C during List 2 learning (Tables 15 and 16). Significant differences were found

between all S groups. Poor premorbid schizophrenic Ss made more intrusion errors than did good premorbid Ss. Reactive schizophrenic Ss, in turn, made significantly more errors than did normal Ss. Again, schizophrenic Ss perseverated List 1 response terms, and it was the process Ss' performance on List 2 that was most disrupted by this failure of response inhibition. A significant interaction of degree of original learning and pathology was also found. Schizophrenic Ss perseverated more with overlearning than without, and this difference was significantly greater than the tendency for normal Ss to make more intrusion errors without overlearning. Thus, overlearning made the inhibition of List 1 response terms more difficult for schizophrenic Ss. This finding was congruent with the results of List 1 MMFR.

Returning to a consideration of List 2 errors, good evidence seemed to exist supporting the conception that process schizophrenic Ss failed to inhibit List 1 response terms in the first stage of A-C learning on the second list. That the same mechanism should account for the process schizophrenic deficit seen in the A-Br paradigm seemed reasonable. Unfortunately direct evidence, such as the intrusions into List 2 learning analyzed for A-C, was not available in this paradigm. However, should this supposition have been correct, then the equally poor performance of both schizophrenic groups in paradigm A-Br would have resulted from different types of deficit. Reactive schizophrenic Ss were disrupted by response competition and its

consequent associative interference (Goulet, 1968; Underwood, Runquist, and Schulz, 1959) and by the interference with specific associations characteristic of paradigm A-Br (Postman and Stark, 1969). General evidence from the present study concerning the nature of A-Br interference was cited earlier.

Further evidence in support of this conception was found in the analysis of List 2 MMFR difference scores (lenient scoring minus strict scoring). The data and analysis appear in Tables 13 and 14.

A significant type of interference x degree of original learning x pathology interaction was found. Schizophrenic Ss showed more loss of specific associations in paradigm A-Br than did normal Ss with overlearning, and this difference was significantly greater than that found between the groups in A-C. Without overlearning, schizophrenic Ss still showed slightly greater loss of specific associations in paradigm A-Br than did normal Ss, but in this case the difference between the two groups was significantly greater in paradigm A-C. The triple interaction involving the schizophrenic groups further emphasized differences in the A-Br paradigm although these differences only partially supported the hypothesis put forth (i.e., that loss of specific associations would be greatest for reactive Ss in the A-Br paradigm). With overlearning, reactive schizophrenic Ss showed more loss of specific associations in the A-Br paradigm than did process Ss, and this

difference was significantly greater than that found in the A-C paradigm. However, without overlearning poor premorbid Ss showed more loss of specific associations in A-Br than did good premorbid Ss, and this difference was significantly greater than the difference between the two groups in paradigm A-C.

In summary, the data from the present study provided firm evidence that process schizophrenic Ss perseverate old verbal S-R relationships into new learning where success specifically requires the inhibition of these very relationships. It is also clear that reactive Ss are not affected in the same manner to as great an extent. A reasonable explanation of the equal deficit of these groups in the learning of a new verbal task requiring the recombination of old stimuli and responses appears to be that process Ss perseverate the old combination of stimulus and response, while reactive Ss are disrupted by the more usual associative interference resulting from response competition and loss of specific associations connected with negative transfer in this type of task.

The hypothesis upon which this study was based appears to have been supported. Reactive schizophrenic Ss showed greater performance deficit with increasing associative interference in a task than did process schizophrenic Ss and normal Ss. However, the hypothesis did not anticipate finding the process S performance deficit based on response perseveration.

The results of this study are not completely concordant with the results of the only comparable study in the literature (i.e., Kausler et al., 1964). In terms of absolute transfer that investigation found a significant difference between schizophrenic Ss and normal Ss only in the A-Br paradigm. The use of chronic schizophrenic Ss makes this result all the more confusing. Length of hospitalization for these Ss and the criteria for chronicity are unknown. Kausler et al. did note that the Ss were in partial remission. The Ss in the present study, by contrast, were acute (maximum current hospitalization of one year). The overwhelming majority of these Ss participated in the experiment within three weeks of admission. Thus, many Ss, and particularly process Ss, were still quite disturbed at the time that they were seen. It may be that the process Ss in the current study were manifesting the cognitive style most typical of acutely disturbed process (chronic) schizophrenia, while the Kausler et al. Ss may have returned in part to a cognitive structure characteristic of a less disrupted state (Weiner, 1966).

The present research was stimulated in part by Mednick's (1958) high-drive theory of schizophrenia. What are the implications of this study's findings for that theory? If the good-poor premorbid concept is substituted for the acute-chronic distinction of Mednick, the results can be seen as supporting the Mednickian hypothesis. Mednick predicted schizophrenic performance decrement with

increasing task complexity on the assumption that high drive would interfere with response integration and production in such tasks. Although he was not clear about it, this hypothesis would seem to hold more for acute than chronic schizophrenics, because of the drive reduction found in chronic schizophrenics brought about by tangential, drive reducing thoughts. However, Mednick (1958) did note that:

> ...even the chronic patient is in one sense a very anxious person. He has never had the opportunity to extinguish his prepsychotic fears. They are still elicitable; all that is required is that one break through the schizophrenic's "associative curtains (p. 324)."

The reactive schizophrenics in this study showed increasing performance decrement as task complexity increased from A-C to the A-Br paradigm. This was the result of increasing associative interference consequent to response competition, and in A-Br to disruption of specific associations. The process schizophrenics, on the other hand, reacted to increasing task complexity in a manner in keeping with a lower drive state, i.e., they simply perseverated old learning. Both of these findings appear to be supportive of Mednick's conception.

DeWolfe (1971) has elaborated a theory of schizophrenic cognitive deficit based on modified Hullian drive notions. His conceptualization is similar in some respects to Mednick's theory. He explained the schizophrenics' motivational state in terms of available coping energy (ACE). This is energy available to the individual for adaptive functioning and not so undirected with respect to

stimulus as to be disruptive, nor so invested in defense as to be unavailable. DeWolfe hypothesized an inverted-U relationship between ACE and emotional expression. He further refined his theory by noting that the differing premorbid life experiences of process and reactive schizophrenics lead to different cognitive structures in the two groups. He related ACE and cognitive structure to task performance.

The results of the current study are compatible with DeWolfe's theory. Process schizophrenics were described by DeWolfe as performing poorly at all levels of external stress because of their history of reduced consensual perception and cognitive function and low energy available for task relevant behavior. However, increased external stimulation may improve their performance somewhat in that the arousal increment may increase their level of ACE. In the present study, the process Ss performed adequately in the C-D paradigm (a simple task). However, their performance showed a decrement in paradigms A-C and A-Br where the presence of associative interference called for a high level of ACE to adequately meet the task. In these paradigms ACE may have actually been reduced for process Ss by withdrawal of affect. The remaining ACE may have been channeled into the least energy demanding behavior, i.e., perseveration of List 1 responses. In fact, it is possible that process Ss selectively withdrew ACE investment in A-Br and A-C and used it in the C-D paradigm (the easiest task) to produce an adequate performance.

DeWolfe predicted different cognitive deficits under low and high stress for good premorbid schizophrenics. He expected that under low stress and with simple tasks reactive schizophrenics would show performance superior to process schizophrenics and similar to that of normals. This prediction was based on the relatively normal cognitive development and social functioning of these individuals during the premorbid period. With low external stress less emotional expression was expected with a concommitant increase in ACE. In the A-C paradigm with intermediate associative interference and possibly intermediate arousal, reactive Ss showed negative transfer, but their performance was more similar to the normal Ss than to that of process Ss. However, in paradigm A-Br reactive Ss were disabled much more than normal Ss and were similar to process Ss. DeWolfe's theory would predict this good premorbid performance deficit because of extreme emotional reactivity under high external stress (i.e., the great interference in the task) which would reduce ACE. Thus, DeWolfe's theory with its emphasis on a drive-stimulus (S_D) formulation rather than on a generalized drive (D) conceptualization accounts well for the current results.

A fairly simple explanation of the results of this study may be found implicitly in Mednick's conceptualization and in the theorizing of DeWolfe. The increasing disruption of reactive schizophrenics with increasing task complexity is the result of the interaction of high drive and associa-

tive interference. Process schizophrenics have learned to reduce drive by thinking tangential thoughts (Mednick) and by social and emotional withdrawal (DeWolfe). The present study indicates that process schizophrenics may protect (defend) against drive increases by repeating the task relevant behaviors of least drive-evoking nature, i.e., in the face of associative interference they perseverate old, simple, less drive elevating List 1 stimulus-response relationships. Reactive schizophrenics, on the other hand, have not learned this defensive response because their premorbid life experience has not required it. Therefore they are subject not only to performance disruption but probably to some reciprocal augmentation of drive.

One final, possible explanation of the findings should be mentioned. Although the use of the term "perseveration" to describe the behavior of process schizophrenics in this study need not imply the existence of intra-cranial organic pathology in this group, this is an explanation which some would put forth (e.g., Belmont, Birch, Klein, and Pollack, 1964). Reactive schizophrenics in this context might be disrupted by the interaction of their drive state (psychogenic) and task complexity. The process Ss, however, respond to task complexity by perseverative behavior which belies their underlying neurological damage.

While several explanations of the current findings are possible, those incorporating aspects of drive theory seem most acceptable. Mednick's theory appears to be most

parsimonious not requiring the additional concept of ACE needed by DeWolfe's formulation. However, some existing process-reactive literature in other areas appears to be best explained by the use of this concept. Further empirical test should help to ascertain whether or not ACE is a necessary concept. The results of the present study hopefully will serve as impetus to reopen investigation in the area of associative processes and interference in schizophrenia. Not only will such research possibly contribute to an answer for the theoretical question raised above, but may also lead to investigations concerning differential therapeutic interventions in schizophrenia. The difference found in the nature of the verbal learning deficit in good and poor premorbid schizophrenics points to the need for such a discrimination.

Summary

A mixed list (paradigms A-Br, A-C, and C-D) verbal learning technique was used to study the effects of increasing associative interference in tasks upon the performance of process and reactive schizophrenic and normal Ss. The experimental manipulation of interference was found to be effective and produced negative transfer in A-Br and A-C for all Ss. The schizophrenic Ss, as a group, showed significantly greater performance decrement in paradigms causing associative interference than did normal Ss. Process Ss were equally disrupted in paradigms A-Br and A-C. Reactive Ss were disrupted to an extent equal to that of process Ss in A-Br but showed less performance decrement in A-C. Analysis of paradigm specific List 1 response intrusions into List 2 learning in A-C showed process Ss perseverating List 1 responses to a significantly greater extent than reactive Ss who in turn perseverated such responses more than normal Ss. The findings were seen as reflecting reactive schizophrenic performance disruption under conditions of negative transfer as resulting from associative interference based on response competition and the loss of specific associations. Process Ss were seen as responding to the same interference factors by perseveration of old learning. The implications for several theories of schizophrenic etiology, including Mednick's high-drive theory, were discussed.

Appendix

Experimental Lists

List 1 pairs	List 2 pairs
King-Music	King-Rough
Eagle-Rough	Eagle-Music
Baby-Long	Baby-Anger
Table-Fruit	Table-Health
Justice-Quiet	Priest-Window
Dream-Green	City-Slow

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APPROVAL SHEET

The Dissertation submitted by Clifford L. Hirsch has been read and approved by members of the Department of Psychology.

The final copies have been examined by the director of the Dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the Dissertation is now given final approval with reference to content and form.

The Dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

May 17, 1971
Date

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Signature of Advisor